EXHIBIT 17

W. R. Grace & Co. Bankruptcy

Supplemental/Rebuttal Report on Asbestos and Disease Causation

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July 31, 2007

Dose-response relationships for asbestos and mesothelioma are based on observations with cumulative exposures of more than 15 f/ml-y.
As I have said in an earlier report, the relationship between dose of asbestos and the risk of mesothelioma has been derived from observations in occupational cohorts with high cumulative exposure to asbestos. Average cumulative exposure in these cohorts is greater than about 15 f/ml-y. Although a number of case-control studies have reported associations between work in specific industries and in specific jobs and malignant mesothelioma, these studies have no direct or reliable information on the actual level of exposure. The dose of asbestos at which a doubling of risk occurs has to be extrapolated from direct observations at higher levels of exposure using a mathematical formula, and depends on fiber type.

In evaluating the role of any specific exposure in causing mesothelioma, all other exposures to asbestos and possible high-dose radiation should also be considered.
This conclusion would appear to be self-evident. I had suggested in my supplemental report that estimation of attributable fraction for specific exposures be based on the Peto formula as extended by Price & Ware (2005) when sufficient information is available, and on the formulas in Hodgson & Darnton (2000) otherwise. Dr. Roggli disagrees saying that there are strong biological arguments against using the Peto formula for multiple exposures to asbestos. He again attributes to me an argument that I did not make, saying, "[f]inally, he refers to the Peto model as indicating that earlier exposures have a greater attributable risk to (sic) mesothelioma than later exposures in an individual case." I said absolutely nothing of the sort. Dr. Roggli appears to be rather confused about the proper use of the Peto formula. The Peto formula is a description, based loosely on the idea of multistage carcinogenesis, of the incidence of mesothelioma in a population exposed to asbestos. It is only tangentially related to latency (which is a nebulous and poorly defined concept to begin with). Dr. Roggli appears to be quite unaware of the very large body of literature on the application of multistage models to situations in which there are multiple time-dependent exposures to multiple agents. In particular, he appears to be unaware that Price & Ware have extended the Peto formula to the situation in which multiple exposures to asbestos can occur.

The most important point that claimants' experts fail to address is that statements asserting that specific exposures contribute substantially to disease are essentially quantitative statements. Such statements must be evaluated within the framework of a dose-response relationship for the disease, which includes a consideration of the level of exposure actually observed to be significantly associated with the disease, and all other exposures that may contribute to the risk of the disease. Instead of such a critical evaluation, claimants' experts rely on blanket statements, with no supporting evidence, regarding the absence of a threshold.

B. Lung Cancer

Cigarette smoking is the most important cause of lung cancer.

By far the single most important cause of lung cancer is cigarette smoking. The risk depends on both number of cigarettes smoked per day and duration of smoking. Among heavy smokers relative risks can be 20 or higher. There are, in addition, a number of other causes of lung cancer such as ionizing radiation, arsenic, and asbestos.

Lung cancer can occur spontaneously.
Like mesothelioma, lung cancer also occurs spontaneously. Based on a large cohort study, a recent publication (Thun et al., 2006) estimates that the age-standardized lung cancer mortality rates among non-smoking men and women are 171 and 147 per million person years, respectively. These rates can be taken to be reasonable approximations to the spontaneous rates of lung cancer, which are, therefore, considerably higher than the spontaneous rates of mesothelioma, which are between 2 and 4 per million person years as I have said above.

Not every exposure to asbestos is a significant contributing factor to lung cancer.
The argument here is essentially the same as the argument I have made for mesothelioma above and in previous reports.

A doubling of the risk of lung cancer occurs at high exposures to asbestos, more than 100 f/ml - yr.
As I discussed in my supplemental report, EPA uses a potency factor of $K_L = 0.01$ in estimating the Unit Risk of asbestos exposure in its IRIS database. The 2004 analysis (McDonald et al., 2004) of the Libby miners' data estimates $K_L = 0.0036$ for Libby fibers. Using the standard linear relative excess risk model used by EPA for lung cancer these estimates of K_L imply a doubling of lung cancer risks at cumulative exposures of 100 and 278 f/ml-y, respectively. Thus, even the EPA estimate of K_L, which likely overstates the true risk of lung cancer from asbestos exposure, implies a doubling of risk only at very high cumulative exposure. A meta-analysis of asbestos exposure and lung cancer by Goodman et al. (1999) concludes that asbestos-associated lung cancer risk is highly variable in the 69 cohorts examined. Moreover, Goodman et al. report that the RRs in most cohorts were below 2 suggesting that very high exposures to asbestos are required for a doubling of lung cancer risk. As is the case for mesothelioma, the dose-response relationship is derived from observations in cohorts of occupationally exposed workers with average exposures greater than about 15 f/ml-y.

In her report, Dr. Welch contends that the re-analyses of the Libby data by Sullivan et al. (2007) shows that the data are consistent with the conclusion that a cumulative exposure as low 4 f/ml-yr[7] is consistent with a doubling of the risk for lung cancer because the 95% confidence interval for the standardized mortality ratio (SMR) includes 2. She should note also that the 95% confidence interval includes 1 and, therefore, this cumulative exposure is equally consistent with absolutely no increased risk of lung cancer. In fact, that is the usual and proper interpretation of such a result taken in isolation: the result is not statistically significant and therefore the data are consistent with no increase in risk. However, a complete evaluation of this result requires that it be

[7] Throughout her expert report Dr. Welch reports cumulative exposures in units of fibers/year. I assume she means fibers/ml x years = f/ml-y.

considered within the general framework of the full lung cancer analysis presented by Sullivan et al. (2007). These results are presented in table 3 of Sullivan et al. The second to last column shows the SMR increasing from 1.5 for cumulative exposures less than 4.5 f/ml-y to 1.9 for cumulative exposures of greater than 100 f/ml-y, an extremely shallow dose-response relationship. Such a shallow dose-response relationship is totally inconsistent with a doubling of risk at less than 4 f/ml-y cumulative exposure. The last column of the same table shows the standardized rate ratios (SRR), which is an estimate of the risk relative to the lowest exposure group (less than 4 f/ml-y). Like the SMR, the dose-response relationship for the SRR is extremely shallow and, in fact, the SRR for the highest exposure group (greater than 100 f/ml-y) is only 1.5 and not statistically significant. It is again inconceivable that a doubling of risk would occur for a cumulative exposure less than 4 f/ml-y, but that the risk for a cumulative exposure of greater 100 f/ml-y would be only 1.5 times the risk of a cumulative exposure less than 4 f/ml-y. It is hard to believe that Dr. Welch is not aware of these fundamental tenets of epidemiology.

Had Dr. Welch done a critical review of the Sullivan paper, she would have noted also that the paper reported some extremely strange results that cast doubt on all the findings. For example, in the footnote to table 3, Sullivan et al. report that the slope for the lung cancer exposure response relationship is estimated to be 5.479×10^{-6}, which is far too small to describe the pattern of standardized rate ratios (SRR) shown in that table. A similar comment can be made for the reported slope for non-malignant respiratory disease. Despite the limitations of the Sullivan analyses, it is clear from table 3 of that paper that a doubling of lung cancer risk at Libby requires a cumulative exposure greater than 100 f/ml-y. As I have pointed out in my earlier reports, even the EPA slope factor for lung cancer, which is likely to be conservative, implies that a doubling of lung cancer risk occurs at a cumulative exposure of about 100 f/ml-y. The McDonald et al. (2004) analysis of the Libby data indicates that a doubling of lung cancer risk at Libby requires a cumulative exposure of about 278 f/ml-y. I note also that the original NIOSH study conducted by Amandus et al. (1987) reported a lung cancer slope factor of 0.58 per 100 f/ml-y cumulative exposure at Libby. This estimate translates to a doubling of risk at a cumulative exposure of about 172 f/ml-y. I also pointed out in my earlier reports that a meta-analysis conducted by Goodman et al. (1999) implied that a doubling of risk occurs at very high cumulative exposures to asbestos.

Thus, the available literature, both on asbestos generally, and Libby fibers specifically, clearly indicates that a doubling of lung cancer risk occurs only at substantial cumulative exposures, above 100 f/ml-y.

In evaluating the role of any specific asbestos exposure in causing lung cancer, exposure to smoking and all other exposures to asbestos must be considered in addition to exposure to the myriad other causes of lung cancer.

In my supplemental report I have presented a quantitative analysis of the risk associated with joint exposure to asbestos and cigarette smoking and made suggestions regarding the estimation of the fraction of risk attributable to asbestos exposure. Specifically, since smoking is by far the strongest risk factor for lung cancer, in any lung cancer case, the contribution made by smoking must be evaluated before any additional risk conferred by